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FAT EMBOLISM SYNDROME, IN A PATIENT WITH BILATERAL TOTAL KNEE REPLACEMENT

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ABSTRACT:

BACKGROUND:
Fat embolism syndrome is caused by the release of fat globules into the systemic circulation. The presentation varies from mild respiratory distress to a devastating clinical scenario that includes hypoxemia, petechial hemorrhages of the skin, and confusion that may lead to coma or death.It is prevalent in 1-5% of patients after trauma to the pelvis, long bones and rarely described in elective total knee replacement.

CASE DESCRIPTION:
We describe a case of fat embolism syndrome after elective bilateral total knee replacement with cerebral and pulmonary involvement, showing typical imaging findings in brain magnetic resonance imaging and pulmonary computed tomography.

CONCLUSION:
Fat embolism syndrome is a rare complication of bilateral total knee replacement.

KEY WORDS:
Fat embolism syndrome, Total knee replacement, Magnetic resonance image.

INTRODUCTION:

Fat embolism is a common complication after long bone fractures. Fat embolism results in the fat embolism syndrome,which is associated with multiorgan dysfunction involving the lung, brain and skin due to entry of fat globules into the systemic circulation.Fat embolism syndrome typically occurs 24 to 72 hours after the initial injury and usually appears with a classical triad consisting of hypoxemia, neurological abnormalities and petechial rashes.The treatment of FES is largely supportive with adequate oxygenation, early resuscitation, minimizing the stress response and hypovolemia. We presented a case of fat embolism syndrome with cerebral and pulmonary manifestation, as a rare complication of elective bilateral total knee replacement.

CASE REPORT:
A 65 year old lady known diabetic and hypertensive with history of bilateral knee pain for 8 years and restricted mobility for 2 months, was evaluated for knee pain and a diagnosis of degenerative osteoarthritis was made. She was admitted electively for bilateral total knee replacement.Pre-operative workup was done including, complete blood picture, Urea, Creatinine, Electrolytes, Liver function tests, Electrocardiogram and Echocardiography allof which were within normal limits. She was planned for bilateral total knee replacement. Procedure was performed under spinal anesthesia and an epidural catheter was inserted into the sub-arachnoid space, 10 mg of bupivacaine was injected, surgery was started after block confirmation. Knees were approached through mid-line incision, titanium-implants were placed and after re-surfacing sutures were applied. During surgery she remained vitally stable, maintained oxygen saturation upto 96% on 2-3litres of oxygen. Total duration of surgery was 3 hours with total anesthesia for 4 hours. Estimated blood loss was 1 liter and 1 packed red blood cells was transfused post-operatively. After confirmation of return of sensation, patient was transferred to the recovery room. Pain was controlledby fentanyl through the epidural catheter. She remained vitally

stable. Patient was shifted to the intensive care unit for post operative monitoring. After 8 hours she was found to be drowsy and having new onset of right sided weakness. Her sedation was stopped and she was re-evaluated by the neurology team. On examination she was drowsy, grimacing on pain stimulus, no facial asymmetry was seen, she was localizing from the left upper limb and withdrawing from the left lower limb, and theplantars were equivocal.

Her MRI Brain was done which showed diffusion restriction in multiple areas including Right cerebellar, left periventricular and centrum semiovale (Fig 1 a, b& c)

She was started onAspirin 75 mg and Enoxaprin 40mg subcutaneously daily for DVT prophylaxis.She regained consciousness after 4 days and was able to follow single step commands she was then shifted to the ward. Where the next morning, she became tachypenic, tachycardiac and her ABGs(arterial blood gases) showed respiratory alkalosis so an urgent CTPA(CT pulmonary angiogram) was done which showed partial thrombosis in peripheral branches of pulmonary vasculature.(Fig 2)Diagnosis of Fat embolism syndrome was made.She received Dexamethasone 30 mg for 48 hours.Her oxygen saturation was maintained upto 96% and she was transfused 2 packed red blood cells. Patient improved symptomatically after 72 hours and her rehabilitation was started. Pulmonology consultation was done and they defer therapeutic anticoagulation due to partial thrombosis in the peripheral branches and patient clinical improvement after few hours favors spontaneous recanalization.

DISCUSSION:

Fat embolism syndrome is caused by the release of fat globules into the systemic circulation. The presentation varies from mild respiratory distress to a devastating clinical scenario that includes hypoxemia, petechial hemorrhages of the skin, and confusion that may lead to coma or death.It is prevalent in 1-5% of patients after trauma to the pelvis, long bones and rarely described in elective total knee replacement.Lee et alretrospectively reviewed 2345 patients with bilateral total knee replacement and found that the incidence of fat embolism syndrome with cerebral manifestations was 0.0.17%. [1]
Gurd and Wilson have suggested that at least two major criteria or one major plus 4 minor criteria. Major criteria include cerebral involvement, respiratory insufficiency and petechial rash. Minor include fever, tachycardia, jaundice, deranged renal parameters, retinal changes, anemia, thrombocytopenia, raised ESR and fat macroglobulinemia (fat particles in blood). [4]

Entry of fat globules into venous circulation is through 2 mechanisms, either by entrance of fat droplets into the disrupted veins in the vicinity of the trauma due to the pressure difference between the venous pressure and intramedullary pressure. Or the traumatic injury to bone results in release of fat droplets into the venous circulation, then from veins to the heart. In the heart due to presence of septal defects either a Patent foramen ovale or Atrial septal defects or less likely the ventricular septal defect, the fat globule travel from the heart into systemic circulation and then to the brain or lungs. In the brain, these fat globules produce inflammation in the walls capillaries. The inflammation causes chemical changes that may cause clumping of platelets, which results in decreased circulation to the areas of the brain fed by those capillaries. Another biochemical theory is hydrolyzation of fat due totrauma to the bone induces chemical changes that causes release of free fatty acids into the body. These fatty acids causes stagnation of blood and results in decreased circulation. Last, is the activation of complement system and factor VII, due to thromboplastin release, which leads to intravascular coagulation and contributes to the non-traumatic cases of Fat embolism syndrome.[6]

Neuroimaging accurately depicts the cerebral change in FES.These lesions are micro-infarction due to microscopic fat embolisms. Diffusion-weighted and susceptibility weighted imaging sequences are the mainstay of imaging sequences which usually showed diffusion restriction in the cerebral deep white matter typically in the region of the centrum semiovale and corona radiate, as seen in our case. These MRI findings along with the clinical scenario were associated with high accuracy in making the diagnosis of cerebral FES.[3]

Three predominate patterns observed with pulmonary involvement in fat embolism syndrome are ground-glass change with geographic distribution, ground glass opacities with interlobular septal thickening, and nodular opacities with no zone predominance or gravity dependence. Our case demonstrate filling defects in peripheral pulmonary vasculature. Filling defects in pulmonary arteries are rarely described in non-fulminant fat embolism syndromes.

Literature review showed that modification in surgical techniques or computer-assisted navigation without breaching of the femoral medullary canal has not been associated with a lower incidence of FES.Similarly no difference in incidence of FES was found in patients with unilateral or bilateral TKR.[2]

The treatment of FES is supportive with adequate oxygenation and mechanical ventilation, early resuscitation and minimizing the stress response and hypovolemia. Although our patient received steroids but the use of prophylactic steroid therapy has been considered with limited results and no universal agreement regarding its role.[5]

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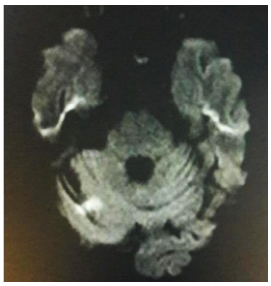
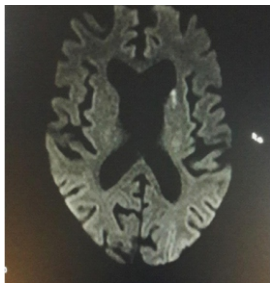


(a) Diffusion restriction in right cerebellar hemisphere



(b) Diffusion restriction Left periventricular region

(c)Diffusion restriction in right & left centrum semiovale, right posterior frontal region

Figure 1: MRI BRAIN

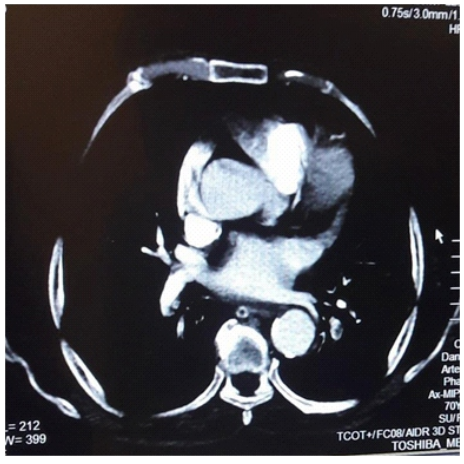


Figure 2:
CT pulmonary angiography
Partial thrombosis of left pulmonary artery

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Author's contribution:
Syeda Saba Zaidi; , concept, data collection, data analysis, manuscript writing, manuscript review
Noman Ishaque; data collection, data analysis, manuscript writing, manuscript review